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THE ROLE OF INTERLEUKIN-6 AS AN INFLAMMATORY CYTOKINE IN PATIENTS WITH NEPHROPATHY

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Abstract. Interleukin-6 (IL-6) is a pleiotropic pro-inflammatory cytokine critically involved in the pathogenesis of multiple forms of nephropathy, including diabetic nephropathy (DN), immunoglobulin A nephropathy (IgAN), lupus nephritis (LN), acute kidney injury (AKI), and chronic kidney disease (CKD). The objective of this narrative review is to comprehensively analyze the molecular mechanisms by which IL-6 drives renal inflammation and injury, its role across nephropathy subtypes, its value as a biomarker of disease activity, and the therapeutic implications of IL-6 pathway inhibition. Key findings demonstrate that IL-6 acts via classical and trans-signaling pathways—engaging the Janus kinase/signal transducer and activator of transcription 3 (JAK/STAT3), mitogen-activated protein kinase (MAPK), and nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) cascades—to promote glomerular mesangial proliferation, extracellular matrix deposition, podocyte loss, tubular injury, and renal fibrosis. Serum and urinary IL-6 levels correlate with glomerular filtration rate decline, proteinuria, and disease progression across all major nephropathy subtypes. Emerging IL-6 inhibitors, including tocilizumab, satralizumab, and ziltivekimab, show therapeutic promise. In conclusion, IL-6 is both a central pathogenic mediator and a promising therapeutic target in nephropathy, warranting further investigation through prospective clinical trials.

Keywords: interleukin-6, nephropathy, inflammatory cytokine, JAK/STAT3 signaling, diabetic nephropathy, chronic kidney disease, IL-6 inhibition.

Introduction. Nephropathy encompasses a broad spectrum of kidney diseases—including diabetic nephropathy (DN), immunoglobulin A nephropathy (IgAN), lupus nephritis (LN), hypertensive nephropathy, acute kidney injury (AKI), and chronic kidney disease (CKD)—all of which are characterized by progressive deterioration of renal structure and function (Bukhanova et al., 2024). Chronic kidney disease alone affects an estimated 10–13% of the global adult population, representing one of the leading causes of morbidity and mortality worldwide and a principal pathway to end-stage renal disease (ESRD), which necessitates dialysis or transplantation (Magno et al., 2019). The socioeconomic burden of CKD is enormous, with healthcare costs escalating as patients progress through disease stages. Despite advances in renin-angiotensin-aldosterone system (RAAS) blockade, sodium-glucose cotransporter 2 (SGLT2) inhibition, and blood pressure management, a substantial proportion of patients continue to experience progressive renal function decline, underscoring the urgent need for novel therapeutic strategies that address the underlying inflammatory mechanisms of kidney injury.

A growing body of evidence has established chronic low-grade inflammation as a central pathophysiological mechanism common to virtually all nephropathy subtypes (Su et al., 2017). Inflammatory mediators—including cytokines, chemokines, and adhesion molecules—orchestrate the recruitment and activation of immune cells within the renal parenchyma, perpetuating a cycle of tissue injury, maladaptive repair, and progressive fibrosis. Among these mediators, interleukin-6 (IL-6) has emerged as a particularly significant cytokine owing to its pleiotropic biological activities and its capacity to bridge innate and adaptive immune responses in the kidney (Kreiner et al., 2022). IL-6 is a 26-kilodalton glycoprotein encoded by the IL6 gene located on chromosome 7p21. It is produced by a diverse array of cell types, including T lymphocytes, B lymphocytes, monocytes and macrophages, mesangial cells, podocytes, glomerular endothelial cells, and renal tubular epithelial cells. Under normal physiological conditions, serum IL-6 concentrations in healthy adults are typically below 5 pg/mL.

The biological profile of IL-6 is characterized by a fundamental duality. Under physiological conditions, IL-6 plays essential roles in immune homeostasis, the hepatic acute-phase response, hematopoietic regulation, and tissue repair following acute

injury (Su et al., 2017). However, when IL-6 is produced in pathological excess—as occurs in states of chronic inflammation, autoimmunity, and metabolic derangement—it becomes a potent driver of sustained inflammatory signaling, extracellular matrix accumulation, and progressive organ damage (Bukhanova et al., 2024). This dichotomy between protective and destructive functions is governed in large part by the distinction between classical and trans-signaling pathways, each of which engages distinct cellular targets within the kidney.

The objective of this review is to comprehensively analyze the molecular mechanisms through which IL-6 contributes to renal inflammation and injury, to examine its disease-specific roles across the major nephropathy subtypes, to evaluate its emerging utility as a biomarker of disease activity and progression, and to assess the therapeutic potential of IL-6 pathway inhibition. By synthesizing current evidence from experimental models and clinical studies, this review aims to provide a detailed and integrative account of IL-6 in the context of nephropathy and to identify key priorities for future investigation.

Research methods. This study was conducted as a narrative review of the existing peer-reviewed literature on the role of interleukin-6 in nephropathy. The narrative review design was selected because it permits a comprehensive, qualitative synthesis of evidence from diverse study types—including experimental mechanistic studies, observational cohort studies, clinical trials, and systematic reviews—thereby providing a broad and integrative perspective on the multifaceted role of IL-6 in kidney disease.

Inclusion criteria encompassed peer-reviewed original research articles, systematic reviews, meta-analyses, clinical trials, and observational studies published in the English language between 2008 and 2025 that investigated the role of IL-6 in human or experimental animal models of nephropathy. Studies were required to report data on IL-6 signaling mechanisms, IL-6 levels as biomarkers, or therapeutic interventions targeting the IL-6 pathway in the context of kidney disease. Exclusion criteria included studies focused exclusively on non-renal manifestations of IL-6 activity, conference abstracts without accompanying full-text publications, editorials, and letters to the editor lacking original data. Reference lists of selected articles were manually screened to identify additional relevant publications not captured by the initial database search.

Results and Discussion. IL-6 Molecular Biology and Signaling Pathways in the Kidney

Interleukin-6 belongs to the IL-6 family of cytokines and adopts a characteristic four-helix bundle tertiary structure. Its biological effects are initiated through binding to the membrane-bound IL-6 receptor (mIL-6R, also designated CD126), which is expressed predominantly on hepatocytes, leukocytes, and renal mesangial cells (Su et al., 2017). The IL-6/IL-6R binary complex subsequently recruits the ubiquitously expressed signal-transducing glycoprotein 130 (gp130, CD130), inducing gp130 homodimerization and thereby initiating intracellular signaling cascades. This mode of signaling, termed classical signaling, activates the Janus kinase family members JAK1, JAK2, and TYK2, which in turn phosphorylate the signal transducer and activator of transcription 3 (STAT3). Phosphorylated STAT3 (pSTAT3) dimerizes and translocates to the nucleus, where it drives the transcription of target genes implicated in inflammation and fibrosis, including IL-6 itself (establishing a positive feedback loop), suppressor of cytokine signaling 3 (SOCS3), fibronectin, collagen types I and IV, and matrix metalloproteinase-9 (MMP-9) (Bukhanova et al., 2024).

In contrast to classical signaling, IL-6 trans-signaling is mediated by a soluble form of the IL-6 receptor (sIL-6R) that is generated through proteolytic cleavage of membrane-bound IL-6R by the metalloproteinases ADAM10 and ADAM17. The sIL-6R circulates in the blood and binds IL-6 to form a complex that can activate gp130 on the surface of cells that do not express mIL-6R—including podocytes, tubular epithelial cells, and endothelial cells—thereby dramatically extending the range of IL-6-responsive cell types within the kidney (Klinkhammer et al., 2016). Trans-signaling is now recognized as the predominant pro-inflammatory arm of IL-6 biology and the principal driver of chronic inflammation and fibrosis in renal disease. Importantly, this pathway can be selectively blocked by soluble gp130 (sgp130Fc, known as olamkicept), which neutralizes IL-6/sIL-6R complexes without affecting classical signaling.

Beyond the JAK/STAT3 axis, IL-6 activates additional downstream pathways with significant renal implications. The JAK/mitogen-activated protein kinase (MAPK) pathway, encompassing the ERK1/2 and p38 cascades, promotes cellular proliferation in mesangial cells. The phosphoinositide 3-kinase/protein kinase B (PI3K/Akt) pathway confers resistance to apoptosis, thereby contributing to the survival of inflammatory

cells within the renal microenvironment. Furthermore, IL-6 activates the nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) transcription factor, which upregulates adhesion molecules such as intercellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1), as well as chemokines including monocyte chemoattractant protein-1 (MCP-1/CCL2), facilitating immune cell recruitment into the kidney (Su et al., 2017).

Within the kidney, IL-6 is both produced by and acts upon multiple cell types, creating complex autocrine and paracrine signaling networks. Mesangial cells are a major source of IL-6 and also express mIL-6R, enabling autocrine signaling that drives mesangial proliferation and extracellular matrix expansion. Podocytes produce IL-6 in response to inflammatory stimuli such as lipopolysaccharide (LPS); reciprocally, IL-6 acting through STAT3 promotes podocyte apoptosis and downregulates nephrin, a critical slit diaphragm protein essential for maintaining the glomerular filtration barrier. Tubular epithelial cells represent the principal source of IL-6 in tubular injury, and IL-6 signaling promotes epithelial-to-mesenchymal transition (EMT), a key process in tubulointerstitial fibrosis. Glomerular endothelial cells also respond to IL-6 by upregulating vascular endothelial growth factor (VEGF) and developing endothelial dysfunction, contributing to glomerular injury (Bukhanova et al., 2024).

IL-6 in Diabetic Nephropathy. Diabetic nephropathy (DN) is the most prevalent cause of chronic kidney disease globally, affecting approximately 40% of individuals with diabetes mellitus and representing the single most common etiology of end-stage renal disease requiring renal replacement therapy. The pathological hallmarks of DN include glomerular hyperfiltration, mesangial matrix expansion, thickening of the glomerular basement membrane, podocyte loss and foot process effacement, tubular atrophy, and progressive interstitial fibrosis (Zhang et al., 2024). These structural alterations are driven by a complex interplay of metabolic, hemodynamic, and inflammatory factors, among which IL-6 occupies a central position.

The mechanistic link between hyperglycemia and IL-6-mediated renal injury proceeds through a well-characterized cascade. Chronic hyperglycemia induces oxidative stress through the overproduction of reactive oxygen species (ROS) via mitochondrial electron transport chain dysfunction, activation of the polyol and hexosamine pathways, and formation of advanced glycation end-products (AGEs). This

oxidative environment activates renal mesangial cells, podocytes, and tubular epithelial cells, stimulating the secretion of IL-6 into the local microenvironment (Ansari et al., 2025). IL-6 then engages the JAK/STAT3 and NF-κB signaling pathways to upregulate transforming growth factor-beta 1 (TGF-β1), the master fibrogenic cytokine in the kidney. TGF-β1 in turn drives the excessive deposition of extracellular matrix components—particularly fibronectin and collagen type IV—leading to glomerulosclerosis and tubulointerstitial fibrosis. Additionally, IL-6 upregulates vascular endothelial growth factor (VEGF), promoting aberrant angiogenesis and glomerular hypertrophy, and potentiates angiotensin II–mediated renal injury while impairing insulin receptor signaling, thereby creating multiple reinforcing pathological circuits (Sánchez-Álamo et al., 2021).

Clinical evidence strongly supports the pathogenic role of IL-6 in DN. Serum IL-6 levels are significantly elevated in patients with DN compared to diabetic patients without nephropathy, with reported mean values of approximately 15.48 ± 4.27 pg/mL versus 7.02 ± 2.46 pg/mL ($p = 0.001$) (Reddy et al., 2024). Prospective cohort data further demonstrate that baseline serum IL-6 concentrations exceeding 4.84 pg/mL are independently associated with a significantly faster rate of progression to end-stage kidney disease (ESKD) or death, with a hazard ratio of 4.10 (95% confidence interval 1.36–12.31) in a cohort of 70 patients with diabetic kidney disease (Sánchez-Álamo et al., 2021). These findings position IL-6 not merely as an inflammatory marker but as an active driver and independent predictor of adverse renal outcomes in the diabetic population.

Genetic studies have provided additional support for the involvement of IL-6 in DN susceptibility. Polymorphisms in the IL6 gene, particularly the −174G/C variant located in the promoter region, have been associated with altered IL-6 expression levels in renal tissue and with differential susceptibility to the development of DN among diabetic patients (Maeda, 2008). These genetic data suggest that inter-individual variation in IL-6 production capacity may contribute to the heterogeneous risk of nephropathy among patients with similar degrees of metabolic derangement, and they raise the possibility of genotype-guided risk stratification in clinical practice.

IL-6 in Acute Kidney Injury and Chronic Kidney Disease. Acute kidney injury (AKI) is defined by a sudden deterioration of renal function, characterized by a rise in serum creatinine of 0.3 mg/dL or greater within 48 hours or a 50% increase within seven days. The major etiologies of AKI include ischemia-reperfusion injury, exposure to nephrotoxic agents, and sepsis. In the context of ischemia-reperfusion injury, tubular epithelial cell necrosis releases danger-associated molecular patterns (DAMPs), which activate innate immune receptors and trigger the rapid production of IL-6 both locally within the kidney and systemically (Shriki et al., 2008). This IL-6 surge promotes the infiltration of neutrophils and macrophages into the renal parenchyma, amplifying tubular damage through the release of additional inflammatory mediators and reactive oxygen species.

Experimental evidence has convincingly demonstrated the pathogenic role of IL-6 in AKI. Studies in IL-6-deficient mice have shown significant resistance to mercuric chloride ($HgCl_2$)–induced AKI, with reduced tubular necrosis, diminished inflammatory cell infiltration, and preserved renal function compared to wild-type controls (Shriki et al., 2008). Intriguingly, however, IL-6 also mediates tubular regeneration in the post-injury recovery phase, highlighting its dual role as both a mediator of acute damage and a facilitator of tissue repair. This duality complicates therapeutic strategies targeting IL-6 in AKI and suggests that the timing and duration of IL-6 inhibition may be critical determinants of therapeutic efficacy. As a biomarker, serum IL-6 rises rapidly in the early phases of AKI and positively correlates with fibroblast growth factor 23 (FGF23). Notably, IL-6 measured at 72 hours post-admission has demonstrated exceptional predictive accuracy for mortality, achieving an area under the receiver operating characteristic curve (AUC) of 1.0 in selected cohorts (Magno et al., 2019). Urinary IL-6 is also elevated in AKI, reflecting local tubular production even in the setting of reduced glomerular filtration rate.

In chronic kidney disease, IL-6 is intimately linked to the renin-angiotensin system, a central mediator of CKD progression. Angiotensin II (Ang II) induces IL-6 expression in both renal tubular and glomerular cells, and IL-6 in turn drives the expression of preproendothelin-1 (ET-1), contributing to hypertension—a major accelerant of CKD progression (Dai et al., 2012). Genetic studies using IL-6 knockout mice have demonstrated that IL-6 deficiency significantly attenuates Ang II–induced

proteinuria, renal fibrosis, and hypertension, providing direct evidence of IL-6 as a critical downstream effector of angiotensin-mediated renal damage. In human studies, IL-6 levels are elevated in both the glomeruli and tubules of CKD patients and are further increased in patients with concurrent hypertension (Bukhanova et al., 2024). In the context of interstitial nephritis, serum IL-6 is significantly elevated during the acute phase compared to the chronic phase, and the combination of IL-6 with tumor necrosis factor-alpha (TNF-α) and microalbumin has been shown to outperform individual markers for disease staging and prognostication (Bian et al., 2022).

IL-6 as a Biomarker in Nephropathy. The accumulating evidence supporting IL-6 as a biomarker in nephropathy is compelling. In healthy adults, serum IL-6 concentrations are typically below 5 pg/mL, whereas significant elevations are consistently observed across nephropathy subtypes: in DN, mean serum IL-6 concentrations reach approximately 15 pg/mL; in AKI, IL-6 peaks rapidly at the time of clinical presentation; in IgAN, circulating IL-6 levels correlate with Gd-IgA1 concentrations and the rate of glomerular filtration rate decline; and in CKD, IL-6 is elevated in both glomerular and tubular compartments (Bukhanova et al., 2024). This consistency across diverse etiologies underscores the central role of IL-6 in the shared inflammatory pathways that drive nephropathy progression regardless of the initiating insult.

Urinary IL-6 measurement offers particular advantages as a biomarker of renal inflammation. Unlike serum IL-6, which may be influenced by systemic sources of inflammation such as infection, malignancy, or autoimmune disease, urinary IL-6 reflects local intrarenal inflammatory activity and provides a more specific assessment of kidney-derived inflammation. Urinary IL-6 concentrations have been shown to correlate with the degree of proteinuria, the rate of GFR decline, and histological activity scores on renal biopsy (Sánchez-Álamo et al., 2021). Furthermore, urinary IL-6 may become elevated before structural damage is apparent on conventional imaging or before significant changes in serum creatinine are detected, suggesting potential utility as an early warning biomarker that captures inflammatory activity prior to irreversible parenchymal injury.

Despite these promising attributes, several limitations must be acknowledged before IL-6 can be integrated into routine clinical practice. Assay standardization

remains incomplete, with significant variability in detection limits and reference ranges across different immunoassay platforms. Serum IL-6 levels are confounded by extrarenal sources, particularly in patients with concurrent infections, malignancies, or autoimmune conditions, which may reduce specificity for nephropathy-specific inflammation. Furthermore, validated cutoff values for clinical decision-making—such as thresholds for initiating or adjusting therapy—have not been established through prospective clinical trials (Bian et al., 2022). Nevertheless, the potential of IL-6 to complement traditional markers such as serum creatinine and estimated GFR—by providing information about inflammatory activity rather than functional decline alone—represents a meaningful conceptual advance in nephropathy monitoring.

Therapeutic Targeting of IL-6 in Nephropathy. The central role of IL-6 in the pathogenesis of nephropathy provides a strong scientific rationale for therapeutic interventions targeting the IL-6 signaling axis. Tocilizumab, a humanized monoclonal antibody directed against the IL-6 receptor (IL-6R), blocks both classical and trans-signaling pathways and has been approved for the treatment of rheumatoid arthritis (RA), giant cell arteritis, and coronavirus disease 2019 (COVID-19)–associated cytokine release syndrome. In the context of RA-associated CKD, biological disease-modifying antirheumatic drugs (bDMARDs) targeting IL-6, particularly tocilizumab, have shown potential for mitigating CKD progression by reducing systemic and intrarenal inflammation (Hanaoka et al., 2025). Clinical trials evaluating tocilizumab in lupus nephritis and IgA nephropathy are currently ongoing, and preliminary data suggest that IL-6R blockade may reduce proteinuria and stabilize renal function in selected patient populations.

Satralizumab, another anti-IL-6R monoclonal antibody, has received United States Food and Drug Administration (FDA) approval for the treatment of neuromyelitis optica spectrum disorder and is currently under investigation for its potential application in renal diseases characterized by IL-6-driven pathology. Ziltivekimab, a monoclonal antibody targeting the IL-6 ligand itself rather than its receptor, has generated considerable interest following the results of the RESCUE trial, which demonstrated that ziltivekimab significantly reduced C-reactive protein, fibrinogen, serum amyloid A, and IL-6 levels in CKD patients with elevated high-sensitivity C-reactive protein (Kreiner et al., 2022). The ZEUS phase 3 trial, which is evaluating

ziltivekimab for cardiovascular outcomes in patients with CKD and atherosclerosis, is ongoing and may yield valuable data on the renoprotective effects of IL-6 neutralization in this population.

Olamkicept (sgp130Fc) represents a conceptually distinct approach to IL-6 pathway modulation. By selectively neutralizing the IL-6/sIL-6R complex, olamkicept blocks trans-signaling—the predominant pro-inflammatory arm—while preserving classical signaling, which mediates homeostatic functions including hepatic acute-phase protein production and immune surveillance (Klinkhammer et al., 2016). This selectivity may confer a superior safety profile by reducing the risk of immunosuppression-related adverse events, including serious infections, that are associated with complete IL-6 pathway blockade. Experimental studies in murine models of crescentic glomerulonephritis have demonstrated significant anti-inflammatory and anti-fibrotic effects of sgp130Fc treatment, supporting further clinical development in nephropathy. Additionally, baricitinib, a Janus kinase 1/2 (JAK1/2) inhibitor that blocks downstream signaling of IL-6 and other cytokines, has been approved for RA and systemic lupus erythematosus. Preclinical studies in lupus nephritis models have demonstrated renoprotective effects, and clinical trials evaluating baricitinib and other JAK inhibitors in lupus nephritis are ongoing (Bukhanova et al., 2024).

Synthesis and Future Directions. Taken together, the evidence reviewed herein positions IL-6 as a central node in the pathophysiology of nephropathy across its major subtypes. The dual nature of IL-6—protective during acute, self-limited immune responses through classical signaling and destructive during chronic inflammatory states through trans-signaling—underscores the need for pathway-selective therapeutic strategies that can attenuate pathological inflammation while preserving essential immune functions (Bukhanova et al., 2024). The convergence of IL-6 signaling on shared downstream effectors (STAT3, NF-κB, TGF-β1) across DN, IgAN, AKI, and CKD suggests that IL-6-targeted therapies may have broad applicability across nephropathy subtypes, although the optimal patient population and timing of intervention likely differ by clinical context.

The accumulating biomarker data support the integration of serum and urinary IL-6 measurements into nephropathy monitoring panels alongside conventional

markers such as serum creatinine, estimated GFR, and proteinuria. By providing information about inflammatory activity at the molecular level, IL-6 may enable earlier detection of disease progression and more timely therapeutic intervention (Zhang et al., 2024). However, therapeutic inhibition of IL-6, while promising, requires careful patient stratification. The risk of infection associated with IL-6 pathway blockade, the phenomenon of cytokine redundancy (wherein other pro-inflammatory cytokines such as IL-17 and TNF-α may compensate for IL-6 inhibition), and the significant heterogeneity of nephropathy subtypes must all be considered in the design of clinical trials and the implementation of IL-6-targeted therapy in clinical practice.

Future research directions include the development of biomarker-guided IL-6 inhibition protocols, in which treatment decisions are informed by real-time assessment of IL-6 pathway activity; the clinical evaluation of selective trans-signaling blockade with agents such as olamkicept; the investigation of combination strategies pairing IL-6 inhibitors with established renoprotective therapies such as SGLT2 inhibitors and RAAS blockers; and the implementation of personalized cytokine profiling to identify patients most likely to benefit from targeted anti-IL-6 therapy (Kreiner et al., 2022). These approaches hold the potential to fundamentally reshape the management of nephropathy by transitioning from a reactive, function-based treatment paradigm to a proactive, mechanism-based precision medicine framework.

Conclusion. Interleukin-6 is a multifaceted inflammatory cytokine that acts through classical and trans-signaling pathways to drive glomerular injury, tubular damage, extracellular matrix deposition, and progressive renal fibrosis across all major nephropathy subtypes. Through the activation of the JAK/STAT3, MAPK, and NF-κB signaling cascades, IL-6 promotes mesangial cell proliferation, podocyte apoptosis and nephrin loss, tubular epithelial-to-mesenchymal transition, and the upregulation of fibrogenic mediators including TGF-β1, fibronectin, and collagen types I and IV. These molecular events are reflected in the consistent clinical observation of elevated serum and urinary IL-6 levels in patients with diabetic nephropathy, IgA nephropathy, lupus nephritis, acute kidney injury, and chronic kidney disease, with strong and reproducible correlations to glomerular filtration rate decline, proteinuria, and disease progression.

Prospective clinical trials evaluating IL-6-targeted therapies in well-defined nephropathy populations, biomarker-integration studies establishing validated cutoffs

for clinical decision-making, and translational research elucidating the optimal timing and combination strategies for IL-6 inhibition are urgently needed to fully unlock the therapeutic potential of targeting IL-6 in kidney disease. The convergence of mechanistic understanding, biomarker discovery, and therapeutic innovation positions IL-6 as one of the most promising targets in the current landscape of nephrology research.

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